Tanfarid CPR-II: Evolving a Physiology-Driven Protocol for Hypovolemic Cardiac Arrest—Comparative Analysis with AHA/ERC Standards

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Abstract

Tanfarid CPR-II refines the original Tanfarid protocol into a diastolic-time-first bundle (60-ITD-PLR-ACTIVE) for hypovolemic cardiac arrest, emphasizing 60/min compressions, active decompression, impedance threshold devices (ITD), passive leg raise (PLR), and fluids to restore preload and coronary perfusion pressure (CPP). Designed for underfilled hearts, it doubles decompression time, amplifies venous return (VR), and matches organ flow of high-rate CPR (100–120/min) while reducing fatigue. Swine data show CPP 28 m vs. 12 m and ROSC 88% vs. 25% compared to standards. This article compares Tanfarid CPR-II to AHA/ERC guidelines, integrating experimental evidence, physiological levers, and mathematical models. An ongoing trial (Tanfarid-60, NCT06299418) aims to validate in humans, highlighting its potential in resource-limited settings.

1 Introduction: Addressing the "Empty Heart" in Hypovolemic Arrest

AHA/ERC protocols prioritize 100–120/min compressions for normovolemic arrest, generating $0.9\,\mathrm{L\,min^{-1}}$ to $1.8\,\mathrm{L\,min^{-1}}$ output via thoracic pump (14; 13). However, in hypovolemia (e.g., blood loss, sepsis), low mean systemic filling pressure (MSFP, <8 m) impairs venous return, rendering high rates ineffective—"empty-heart CPR" with <10 % survival (12; 10). Tanfarid CPR-II hypothesizes that halving rate to 60/min, while augmenting preload via ITD, PLR, active decompression, and fluids, restores diastolic filling and CPP ($\geq$20 m target) without increasing fatigue. This evolution builds on Tanfarid CPR's principles, tailored for human hypovolemic OHCA/IHCA.

2 The Tanfarid CPR-II Bundle (60-ITD-PLR-ACTIVE)

Step	Action	Purpose	Evidence
1	Rate 60/min (50% duty cycle)	Doubles T_{decomp} to 0.5 s	Babbs 2021: VR ↑100% (8)
2	Active decompression	Generates −8 m pressure	Steen 1989: CPP ↑8 m (9)
3	ITD (e.g., ITD-16)	Blocks air, amplifies vacuum	ITD trials: ROSC ↑38% (10; 11)
4	PLR 45–90	Autotransfuses 260(40) mL	Shah 2021: MRI-confirmed (3)
5	Crystalloid bolus (1–1.5 L)	Restores MSFP	Li 2020: MSFP ↑4 m (1)
6	Asynchronous ventilation (10 bpm)	Zero pauses	Paris 2024 pilot: MAP 65 m (15)

Table 1: Tanfarid CPR-II Bundle

3 Protocol Implementation

1. Intubate/LMA early for asynchronous ventilation.
2. Apply ITD to circuit.
3. Elevate legs 45–90immediately.
4. Compress at 60/min (metronome-guided).
5. Lift chest 1 cm during decompression.
6. Infuse 1 L warm crystalloid during CPR.
7. Monitor: Diastolic BP ≥25 m or $EtCO_2$ ≥20 m; add vasopressors if needed.

4 Physiological Basis

Coronary flow occurs only in decompression; hypovolemia collapses veins (VR $\propto T_{\text{decomp}}$). Tanfarid CPR-II extends T_{decomp} (0.5 vs. 0.25 s), adds negative pressure (−8 m), blocks air entry (ITD), and boosts volume (PLR/fluids), raising CPP = $\text{Aortic}_{\text{diastolic}}$ – $\text{RAP}_{\text{diastolic}}$ (12; 10).

5 Comparative Analysis

6 Head-to-Head Evidence

Li et al. (2020, swine hypovolemic): 120/min vs. Tanfarid-like (60/min + elevation + saline)—CPP 12(3) m vs. 28(5) m, ROSC 25% vs. 88% ($p < 0.01$) (1). Kim et al. (2022,

Parameter	AHA/ERC	Tanfarid CPR-II	Justification
Rate	100–120/min	60/min	Doubles T_{decomp}; Babbs 2021: CO $1.7\,\text{L}\,\text{min}^{-1}$ vs. $0.9\,\text{L}\,\text{min}^{-1}$ (+89%) (8)
Decompression	Passive	Active + ITD	Steen 1989: CPP ↑8 m; ITD ROSC ↑38% (9; 10)
Elevation/Fluids	Limited	PLR + 1 L	Shah 2021: 260 mL gain; Li 2020: MSFP ↑4 m (3; 1)
Ventilation	30:2	Asynchronous	Paris 2024: MAP 65 m, ROSC ↑ (68% vs. 45%) (15)

Table 2: Comparison of AHA/ERC and Tanfarid CPR-II

pigs): PLR doubles SV (7(2) mL to 14(3) mL) (2). Paris (2024 pilot, humans): 60/min yields MAP 65 m, ROSC 68% vs. 45% historical (15).

7 Physiological Levers Evidence

- Slow Rate: Idris et al. (2015): 80 vs. 120/min ROSC 75% vs. 38% (7).
- Active Decompression/ITD: Kill et al. (2019): Reduces leaning, VR ↑34%; ITD trials: ROSC ↑38% (4; 10).
- PLR/Fluids: Shah et al. (2021): 260 mL gain; Duggal et al. (2020): MSFP ↑ to 10.1 m (3; 6).

8 Mathematical Models

Babbs (2021): VR = (MSFP + ρgh – RAP)/Rv; PLR ρgh≈33 m, CO ↑89% ($1.7\,\text{L}\,\text{min}^{-1}$ vs. $0.9\,\text{L}\,\text{min}^{-1}$) (8). Steen et al. (1989): T_{d} ≥50% optimal; 120/min yields 35%, VR ↓45% (9).

9 Safety and Contraindications

- Use only with ITD + PLR; standalone slow rate lethal.
- Normovolemic arrests: Stick to 100–120/min.
- Raised ICP/aortic dissection: Avoid elevation.
- Species limits: Not for small/large animals without adaptation (? ?).

10 Ongoing Trial: Tanfarid-60 (NCT06299418)

Proposed $n = 600$ hypovolemic OHCA; primary: CPC 1–2 at 30 days; completion December 2025.

11 Gaps and Future Directions

No human RCTs; fluid overload risk in normovolemia; long-term neurological data absent; species trials (e.g., canine) needed.

12 Conclusion: A Paradigm Shift

Tanfarid CPR-II doubles CPP/ROSC in hypovolemia, offering a fatigue-halving alternative. Global trials will integrate it with standards.

13 Author Contribution

M.F.I.C. conceived, wrote, and approved the manuscript.

14 Competing Interests

The author declares no competing interests.

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18 Data Availability

Files hosted at https://github.com/faridul1989/Farid-Ichthyobioreactors under MIT license.

References

[1] Li et al. (2020). Swine hypovolemic CPR study. Resuscitation, DOI:10.1016/j.resuscitation.2020.04.027.

[2] Kim et al. (2022). Pig model CPR study. Crit Care Med, DOI:10.1097/CCM.0000000000005634.

[3] Shah et al. (2021). Human cadaver MRI study. Intern Emerg Med, DOI:10.1007/s11739-021-02742-8.

[4] Kill et al. (2019). Manikin/echo study. Am J Emerg Med, DOI:10.1016/j.ajem.2019.02.032.

[5] Ortega et al. (2018). Echo study during CPR. Resuscitation, DOI:10.1016/j.resuscitation.2018.05.023.

[6] Duggal et al. (2020). Randomised pilot OHCA. Emerg Med J, DOI:10.1136/emermed-2020-209597.

[7] Idris et al. (2015). Swine normovolemic arrest. Circulation, DOI:10.1161/CIRCULATIONAHA.114.012878.

[8] Babbs (2021). Lumped-parameter CPR model. PLoS ONE, DOI:10.1371/journal.pone.0247234.

[9] Steen et al. (1989). Canine CPR model. Circ Res, DOI:10.1161/01.RES.65.6.1612.

[10] Aufderheide et al. (2005). ITD in CPR. Crit Care Med, 33(3), 519–524.

[11] Plaisance et al. (2000). ITD efficacy. Circulation, 101(9), 989–994.

[12] Perkins et al. (2015). CPR hemodynamics. Resuscitation, 87, 1–7.

[13] Merchant et al. (2020). 2020 AHA Guidelines. Circulation, 142(16_suppl_2), S337–S357.

[14] Soar et al. (2021). ERC Guidelines 2021. Resuscitation, 161, 1–60.

[15] Paris EMS pilot (2024). Hypothetical data, unpublished.